ABSTRACT OF DISCLOSURE

The instant invention relates to the determination that constitutively nearly silent GrB locus in human breast carcinoma and osteosarcoma cells activated upon retinoblastoma protein (pRB)-induced growth arrest owing to the usage of an alternative promoter/transcription start site. Cloned novel cDNA from the locus adds 34 amino acid residues to the N-terminus of GrB zymogen. The alternate product has been designated as GrB-NIC. Tumor cells with accumulated endogenous GrB-NIC, whose mature form was identical to lymphocyte GrB but with a distinctive glycosylation pattern, undergoes post-growth-arrest apoptosis that occurs concurrently with pRB cleavage, and are capable of inducing rapid apoptosis of bystander pRB^- tumor cells. Expression of GrB-NIC is also observed in malignant cells of other types as well as in normal non-immune cells upon cell differentiation, especially in differentiating and differentiated neural cells. GrB-NIC plays a physiological role in embryonic, and particularly in early neuronal development. The disclosure further provides compositions and methods utilizing this new GrB-NIC technology.